



NOTES

VENTRICULAR TACHYCARDIA

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Depolarization wavefronts originate in ventricles → ventricles pump > 100 beats per minute ↓ stroke volume
- Premature ventricular contractions (PVCs): single instance of ventricle contracting prematurely
 - ≥ three PVCs consecutively defined as ventricular tachycardia (VT)

TYPES

Monomorphic VT

- Ventricular contractions have typical, uniform shape
- Typical for reentrant circuits
 - Depolarizations begin from same spot, for focal VT because one area of cells in ventricle is responsible
 - Often caused by reentry around scar in ventricular wall; e.g. from previous myocardial infarction (MI)

Polymorphic VT

- > One QRS complex morphology type
 - Includes Torsades de pointes
- Shape of contractions from each beat changes as signal begins in different areas of ventricle
- May occur when pacemaker cells stressed, increasing automaticity rates, including from severe hypoxia

RISK FACTORS

- Ventricular muscle ischemia, structural heart disease, coronary artery disease (CAD), electrolyte abnormalities

COMPLICATIONS

- Sustained VT may result in sudden cardiac death due to insufficient blood perfusion/rapid ventricular fibrillation

SIGNS & SYMPTOMS

- Chest pain, syncope, dizziness, shortness of breath, palpitations

DIAGNOSIS

LAB RESULTS

- Serum electrolytes
- Toxicology studies (therapeutic/recreational drug use)
 - E.g. digoxin, tricyclic antidepressants, methamphetamine, cocaine

OTHER DIAGNOSTICS

ECG

- Determines cardiac rhythm

TREATMENT

MEDICATIONS

- Pharmacotherapy
 - Depending on cause

SURGERY

- Implanted devices

OTHER INTERVENTIONS

- Cardioversion, pacing
- Correct underlying cause

VISIT...

LANZAROTE
Caliente.COM

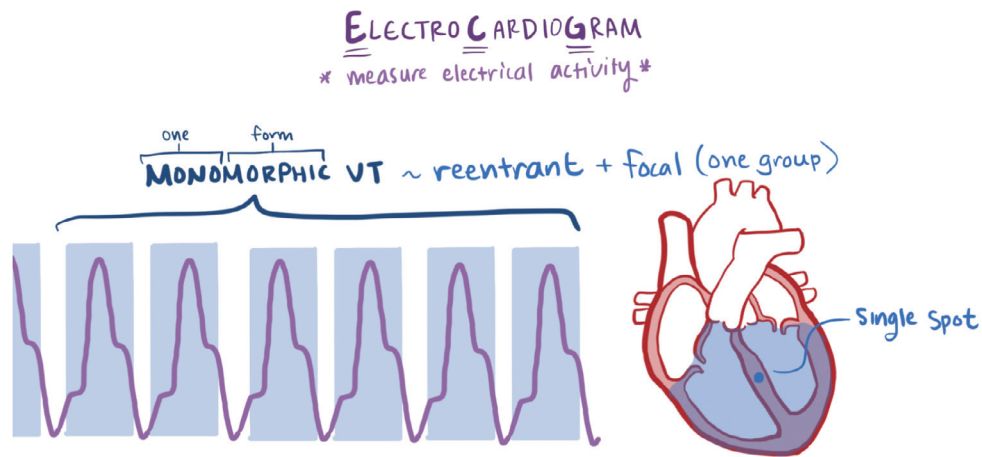


Figure 27.1 Illustration depicting ECG of monomorphic ventricular tachycardia.

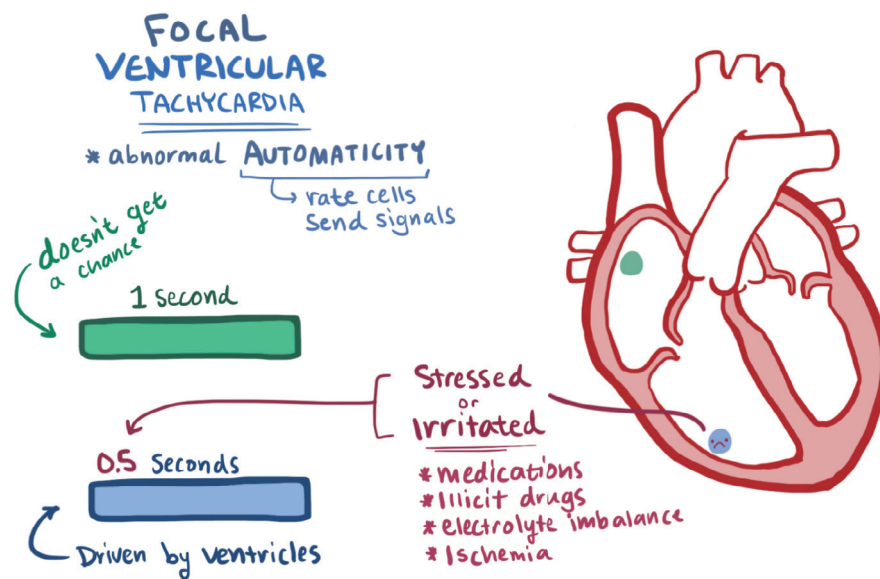


Figure 27.2 Illustration depicting focal ventricular tachycardia.

LONG QT SYNDROME (LQTS)

osms.it/long-qt-syndrome

PATHOLOGY & CAUSES

- Cardiac rhythm disorder characterized by **prolonged ventricular repolarization**
- Characterized by abnormally long QT interval
 - **QT interval**: total time from ventricular depolarization (QRS complex) to complete repolarization (T wave); measured from beginning of QRS to end of T wave
 - QTc (corrected) accounts for changes in heart rate: $QTc = QT \text{ interval} \div \sqrt{RR} \text{ interval (in sec)}$; AKA Bazett formula
 - Adult normal = $420 \pm 20 \text{ msec}$
- Results in ↑ risk of polymorphic ventricular arrhythmias (TdP), which can deteriorate into ventricular fibrillation

TYPES

Inherited

- Caused by **mutations in genes associated with cardiac potassium, sodium channels**
- Triggered by exertion, emotional events, stress, postpartum events, noise
- ≥ 13 types identified; associated with mutations in genes encoding myocyte ion channels
- KCNQ1, KCNH2, KCNE1, KCNE2 affect potassium channels → ↓ outward potassium current
- SCN5A affects sodium channels → ↑ inward sodium current
 - Two distinct LQT phenotypes due to mutant alleles in same locus:
 - **Romano–Ward syndrome**: autosomal dominant; LQTS without hearing loss
 - **Jervell and Lange–Nielsen syndrome**: autosomal recessive; LQTS with congenital sensorineural hearing loss

Acquired

- Usually caused by certain drugs (e.g. anti-infectives, psychotropics, antiarrhythmics, antineoplastics, bronchodilators, gastric motility agents)
 - Common mechanism involves blockage of rapidly activating potassium channels (IKr) current in potassium channel encoded by KCNH2 gene

RISK FACTORS

- Electrolyte imbalances (e.g. **hypokalemia**, **hypomagnesemia**, **hypocalcemia**)
- Underlying heart disease (e.g. HF, hypertrophic left ventricle, history of myocardial infarction)
- Bradyarrhythmias
- Biological females > biological males
- ↑ age
- Inherited genetic mutation
- Postpartum period
 - Related to physiologic stress, altered sleep patterns
- Anorexia nervosa

COMPLICATIONS

- Malignant arrhythmias (**TdP**, **VF**), syncope, seizures, **sudden death**

SIGNS & SYMPTOMS

- Palpitations, lightheadedness, hypotension

DIAGNOSIS

LAB RESULTS

- Serum electrolytes
 - Hypokalemia, hypomagnesemia, hypocalcemia may be present

OTHER DIAGNOSTICS

12-lead ECG

- Prolonged QTc (> 470msec in males, > 480msec in females); presence of tachyarrhythmias (TdP); altered T-wave morphology

Bicycle/treadmill stress test

- Presence of exercise-associated arrhythmias

Catecholamine drug testing

- Differentiates etiology
 - Provocative testing with catecholamine; e.g. epinephrine
 - Measure effect on QT interval

Clinical/family history, physical examination

- With compatible findings

Genetic testing

Schwartz score

- Diagnosis of congenital LQTS by scoring QTc, clinical factors, individual history
- Scoring: probability of congenital LQTS
 - ≤ 1: low
 - 1.5–3: intermediate
 - ≥ 3.5: high

TREATMENT

MEDICATIONS

Congenital LQTS

- Beta-blockers: blunt adrenergic response
- Mexiletine: for sodium-channel mutations
- Flecainide: if SCN5A mutation

Acquired LQTS

- Magnesium sulfate: treatment, prevention of recurrence of long QT-related ventricular ectopic beats
- Isoproterenol: increase sinus rate, decrease QT interval
- Lidocaine/phenytoin: shorten duration of the action potential

SURGERY

Congenital LQTS

- Left cervicothoracic sympathectomy (LCTS), left cardiac sympathetic denervation; implantable cardioverter-defibrillator (ICD); pacemaker

Acquired LQTS

- Pacemaker → if bradycardia triggers arrhythmia

OTHER INTERVENTIONS

Acquired LQTS

- Address underlying cause; e.g. correct electrolyte abnormalities, discontinue offending drug; temporary transvenous overdrive pacing, electrical cardioversion/defibrillation

Lifestyle modifications

- Avoidance of triggering drugs, avoidance adrenergic stimuli; e.g. strenuous exercise, emotional stress

DIAGNOSIS OF CONGENITAL LQTS

CRITERIA	POINTS
ECG FINDINGS	
QTc (ms)	
> 480	3
460-479	2
450-459 (males)	1
4-minute recovery from exercise stress test > 480	1
TdP	2
T wave alternans	1
Notched T wave (in 3 leads)	1
Low HR rate (age adjusted)	0.5

DIAGNOSIS OF CONGENITAL LQTS**CLINICAL HISTORY**

Syncope	
With stress	2
Without stress	1
Congenital deafness	0.5

FAMILY HISTORY

LQTS confirmed in family member	1
Unexplained sudden death in immediate family members (< 30 years of age)	0.5

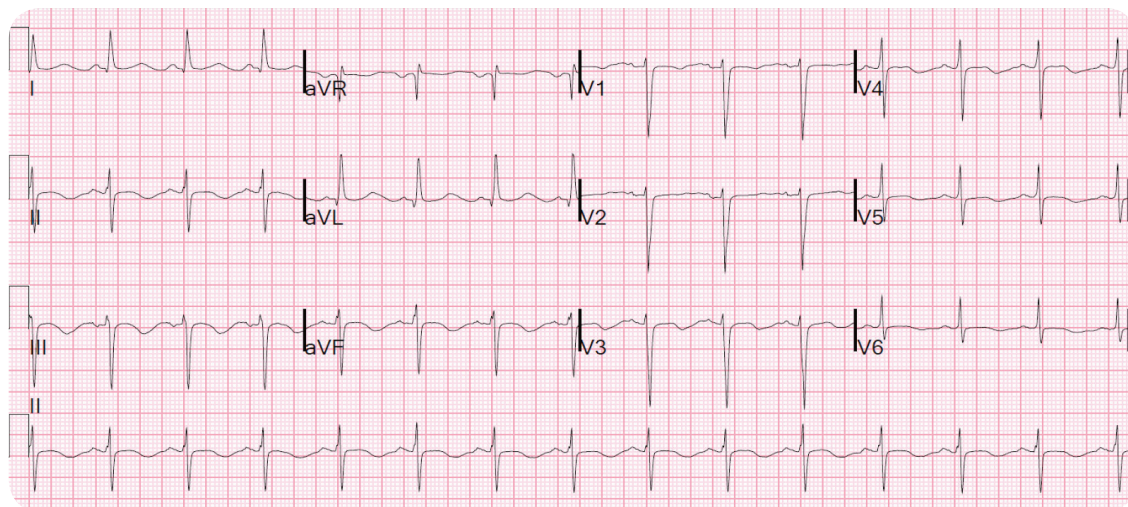


Figure 27.3 ECG trace demonstrating long-QT syndrome.

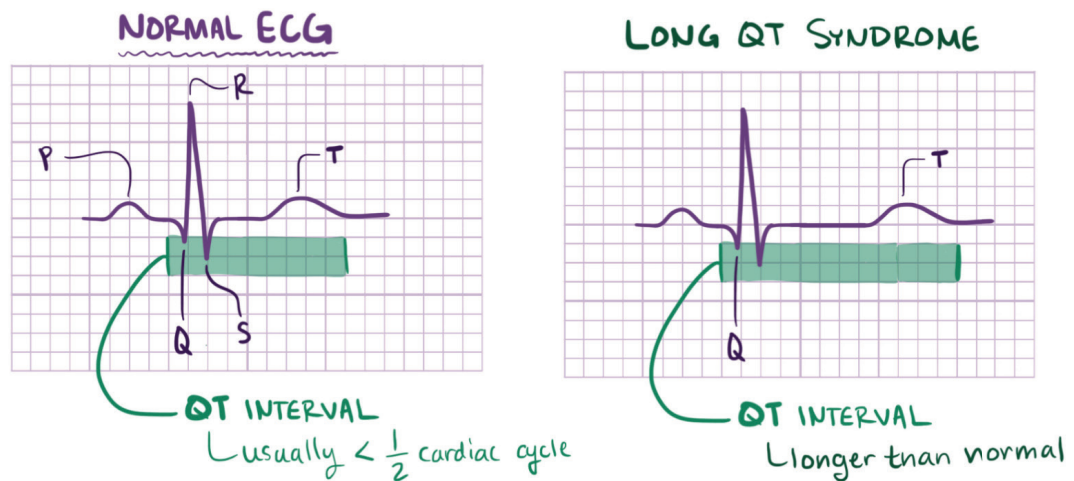


Figure 27.4 ECG trace demonstrating long-QT syndrome.

TORSADES DES POINTES (TdP)

osms.it/torsades-de-pointes

PATHOLOGY & CAUSES

- Literally means “twisting of the points”
- The peaks of QRS complex “twist” around isoelectric line on electrocardiogram
- Lengthening QT interval → early afterdepolarizations (EADs) → premature ventricular depolarizations → polymorphic VT (TdP)
 - May resolve spontaneously
 - Transmural reentry/abnormal automaticity may perpetuate TdP
 - May degenerate into ventricular fibrillation

RISK FACTORS

- LQTS, drugs associated with LQTS
- Bradycardia
- Electrolyte imbalance
- Biologically female
- Anorexia nervosa

COMPLICATIONS

- Ventricular fibrillation, seizures, sudden cardiac death



MNEMONIC: TO4SADE

Drugs that may induce QT prolongation

Thiazides

O4 - Oanzapine, Opioids, Quinidine, Quinolones

Sotalol/SSRIs

Antihistamines/antipsychotics
Anti**D**epressants like TCAs

Erythromycin (Macrolide antibiotics)

SIGNS & SYMPTOMS

- Palpitations, lightheadedness, syncope

DIAGNOSIS

LAB RESULTS

- Serum electrolytes
 - Hypokalemia, hypomagnesemia, hypocalcemia may be present

OTHER DIAGNOSTICS

- 12-lead ECG
 - Ventricular rate: 150–300 beats per minute
 - RR interval: irregular
 - P wave, PR interval: absent
 - QRS duration: > 0.12 seconds; changes amplitude, shape, direction

TREATMENT

MEDICATIONS

- For acquired LQTS/other causes of TdP
 - Magnesium sulfate, isoproterenol, lidocaine, phenytoin
- For congenital LQTS
 - Beta-blockers, mexiletine

SURGERY

- Left cardio-thoracic sympathectomy

OTHER INTERVENTIONS

- Treat underlying cause; e.g. correct electrolyte abnormalities, discontinue offending drug
- Temporary pacing, permanent dual chamber pacemaker, implantable cardioverter-defibrillator (ICD)

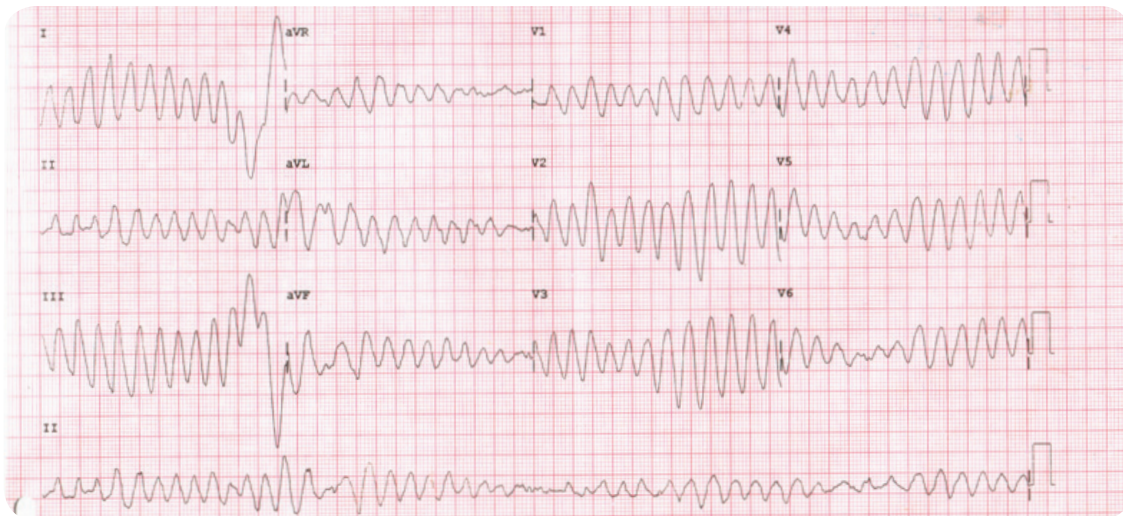


Figure 27.5 ECG demonstrating torsades de pointes.

VENTRICULAR TACHYCARDIA

osms.it/ventricular-tachycardia

PATHOLOGY & CAUSES

- **Ventricular arrhythmia** originating in ectopic ventricular pacemaker, resulting in $\geq$ three premature ventricular complexes (PVCs) occurring at $\geq$ 100 beats/min
- Dysrhythmia may be sustained ($>$ 30 seconds)/nonsustained ($<$ 30 seconds)/paroxysmal
- Abnormally fast ventricular contractions $\rightarrow$ $\downarrow$ ability for ventricles to fill $\rightarrow$ $\downarrow$ **cardiac output** $\rightarrow$ $\downarrow$ perfusion $\rightarrow$ impaired hemodynamics

TYPES

Non-reentrant/focal ventricular tachycardia

- Triggered by abnormal automaticity of specific area of ventricle
 - Ventricular pacemaker cells fire at high rate, preventing pacemaker cells in SA node from firing $\rightarrow$ heart rate driven by ventricular pacemakers
 - May be caused by certain medications, illicit drugs (e.g. methamphetamine, cocaine), electrolyte imbalances, myocardial ischemia in ventricles

Reentrant ventricular tachycardia

- More common than focal VT
- **Reentry**: perpetual electrical signal that occurs due to changes in refractory period length, rate of signal conduction
 - Cardiomyocytes can be altered when stressed/irritated by external stimuli; e.g. medications/illicit drugs: change conduction speed, refractory period
 - Dead cells in myocardial tissue create scar tissue $\rightarrow$ conduction signals go around scar $\rightarrow$ perpetual signal, AKA reentry

RISK FACTORS

- $\uparrow$ age
- Cardiac disease
 - Post-MI, cardiomyopathy, valve disease, HF
- Electrolyte imbalance
- Cardiac ion channelopathies resulting in long QT syndromes
- Infiltrative disease; e.g. amyloidosis
- Pericardial inflammation
- Blunt chest trauma
- Drugs; e.g. cocaine

COMPLICATIONS

- Cardiac ischemia, infarction
- May degenerate into ventricular fibrillation
- Sudden cardiac death

SIGNS & SYMPTOMS

- Chest pain
- Shortness of breath
- Dizziness
- Syncope
- Pallor
- Blood pressure: normal/ $\downarrow$

DIAGNOSIS

LAB RESULTS

- Serum electrolytes
 - Hypokalemia, hypomagnesemia, hypocalcemia may be present

OTHER DIAGNOSTICS

ECG

- **Rate**: $>$ 100 beats per minute, irregular
- **P waves**: may be absent
 - If present, atrioventricular dissociation common (hallmark of VT)

- May be positive/upright or negative/inverted in Lead II
- **PR interval:** none
- **QRS complex:** wide (> 0.12 seconds); ≥ 3 consecutive; distorted shape: may be monomorphic/polymorphic
- **T-waves:** large; polarity may be opposite of major QRS deflection; may be difficult to distinguish

TREATMENT

MEDICATIONS

- Chronic prevention
 - Beta blockers, **amiodarone**, nondihydropyridine calcium channel blockers

OTHER INTERVENTIONS

- Acute treatment
 - Cardioversion
- Drug cardioversion
 - Procainamide, lidocaine, amiodarone frequently used
- Electrical cardioversion
 - Primary treatment for VT associated with hemodynamic instability/when drug cardioversion not immediately available
- Radiofrequency catheter ablation
- ICD